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L3 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1

- AN 1997084811 MEDLINE
- DN PubMed ID: 8931145
- TI Characterization of the S1 binding site of the glutamic acid-specific protease from Streptomyces griseus.
- AU Stennicke H R; Birktoft J J; Breddam K
- CS Carlsberg Laboratory, Department of Chemistry, Copenhagen, Denmark.
- SO Protein science: a publication of the Protein Society, (1996 Nov) Vol. 5, No. 11, pp. 2266-75.
 - Journal code: 9211750. ISSN: 0961-8368.
- CY United States
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 199702
- ED Entered STN: 5 Mar 1997
 Last Updated on STN: 3 Mar 2000
 Entered Medline: 19 Feb 1997
- The glutamic acid-specific protease AΒ from Streptomyces griseus (SGPE) is an 18.4-kDa serine protease with a distinct preference for Glu in the P1 position. Other enzymes characterized by a strong preference for negatively charged residues in the P1 position, e.g., interleukin-1 beta converting enzyme (ICE), use Arg or Lys residues as counterions within the S1 binding site. However, in SGPE, this function is contributed by a His residue (His 213) and two Ser residues (Ser 192 and S216). It is demonstrated that proSGPE is activated autocatalytically and dependent on the presence of a Glu residue in the -1position. Based on this observation, the importance of the individual S1 residues is evaluated considering that enzymes unable to recognize a Glu in the P1 position will not be activated. Among the residues constituting the S1 binding site, it is demonstrated that His 213 and Ser 192 are essential for recognition of Glu in the P1 position, whereas Ser 216 is less important for catalysis out has an influence on stabilization of the ground state. From the three-dimensional structure, it appears that His 213 is linked to two other His residues (His 199 and His 228), forming a His triad extending from the S1 binding site to the back of the enzyme. This hypothesis has been tested by substitution of His 199 and His 228 with other amino acid residues. The catalytic parameters obtained with the mutant enzymes, as well as the pH dependence, do not support this theory; rather, it appears that His 199 is responsible for orienting His 213 and that His 228 has no function associated with the recognition of Glu in P1.